given ample commentary and others are not discussed at all, vascular surgery trainees would be best served by investing the $85.00 cost per copy as an initial installment on *Rutherford's Vascular Surgery* textbook.

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**Mechanoreception by the vascular wall**

Gabor M. Rubanyi. Armonk, N.Y., 1993, Futura, 250 pages, $65.

Physiologists have recognized for nearly a century that the blood vessel is capable of responding to hemodynamic forces. As the flow of blood increases through a vessel, the vessel dilates. This property is intrinsic to the vessel wall and is observed in the absence of neurogenic or hormonal influences. It is now apparent that this flow-mediated vasodilation is caused by the release of vasorelaxant substances from the endothelium. In most vessels the vasodilator that mediates the response to flow is endothelium-derived relaxing factor (EDRF). EDRF is now known to be nitric oxide, derived from the metabolism of arginine by nitric oxide synthase. Another vasodilator that is released from the endothelium in response to flow is prostacyclin, and in some circulations it may even play a larger role than EDRF. Finally, endothelium-dependent hyperpolarizing factors may act on the subjacent vascular smooth muscle to increase the electronegativity of the membrane, thereby reducing intracellular calcium levels and inducing vasorelaxation.

The blood vessel also responds to increase in pressure. As intraluminal pressure increases within a vessel, the vessel contracts. This response appears to be directly generated by the vascular smooth muscle, although it can be modulated by endothelial and neurogenic factors. This myogenic response to elevations in pressure protects the microvasculature from surges in hydrostatic pressure. The myogenic regulation of capillary hydrostatic pressure reduces capillary filtration to about 10% of that which would occur in absence of such regulatory mechanisms.

New insights have been gleaned recently regarding the mechanisms that underlie the vascular response to hemodynamic forces. These insights are reviewed in depth in the text *Mechanoreception by the vascular wall*, edited by G.M. Rubanyi, MD, PhD. The editor is an internationally recognized figure who has made important contributions to this field and is well positioned to provide a seasoned physiologic perspective. The contributors to this textbook are also leading experts in the field who have provided an in-depth review of the biophysical principles of mechanoreception, the phenomena of myogenic tone and autoregulation, and the role of the endothelium in mechanotransduction and vasomotion.

Within this text you will find an elegant discussion of the experimental evidence in favor of different models of the myogenic control of microvascular flow. The modeling becomes quite complex because endothelial and neurogenic influences are superimposed on the myogenic response to elevations in pressure. Furthermore there is a heterogeneity of the strength of the myogenic response, dependent on the vascular bed and size of the vessel. For example, in the cerebral and renal circulation, the myogenic response predominates and plays a major role in maintaining constant flow across a broad range of intravascular pressures. This narrow range of autoregulation is not observed in other vascular beds.

The specific "pressure-sensor" that transduces the hemodynamic stimulus into a mechanical response is still a matter of speculation. Many investigators, however, have calculated that a stretch-responsive calcium channel may be activated by elevations in intravascular pressure. Activation of this calcium channel would increase the influx of extracellular calcium, which would initiate a chain of events leading to vasoconstriction. The evidence that a specific stretch-responsive calcium channel exists is reviewed. Pharmacological data support the contention that a specific stretch-responsive calcium channel can be distinguished from the typical voltage-regulated L-type calcium channels. For example, stretch-induced vasoconstriction is relatively resistant to inhibition by calcium entry antagonists, whereas activation of voltage-regulated calcium channels (by depolarizing concentrations of potassium chloride) are suppressed by these antagonists. Alternatively or in addition, activation of protein kinase C by stretch may enhance the sensitivity of the contractile apparatus to calcium and thereby augment pressure-induced constriction.

The modulatory role of the endothelium receives emphasis in this text. Although the majority of studies indicate that stretch-induced contractile response occurs in the absence of endothelium, there clearly is evidence that the endothelium may modulate pressure-induced vasoconstriction. In the canine basilar artery, stretch-induced release of a vasoconstrictive prostanoid plays a major role in autoregulation. There may be other endothelial constricting factors elaborated through the influence of pressure; the evidence indicates that these may be metabolites of arachidonic acid via the lipoxygenase or cytochrome P450 pathways. Furthermore, increases in pressure can reduce the secretion of endothelium-derived relaxing factor. Finally, in some vascular beds endothelial cells are in direct electrical contact with the vascular smooth muscle through gap junctions. These myoendothelial junctions allow electrical coupling; depolarization of the vascular smooth muscle may lead directly to contraction and indirectly reduce EDRF release.

Nitric oxide also plays a role in inducing vasomotion (the rhythmic contraction and dilation of blood vessels). Nitric oxide–induced increases in guanosine monophosphate may result in oscillation in endothelial calcium or changes in calcium sensitivity of the contractile machinery.

Finally, the intriguing and poorly understood phenomenon of angiogenesis and its regulation by hemodynamic forces is reviewed. Hemodynamic alterations, particularly increases in flow, can initiate angiogenesis. This may be mediated in part by shear stress–induced secretion of tissue

plasminogen activator. Tissue plasminogen activator can activate metalloproteinases which degrade the basement membrane of the endothelial cells. The loss of integrity of the basement membrane allows endothelial cells to migrate through the vessel wall and begin to form new vessels budding off the main channel.

In summary, this is a comprehensive overview of recent insights into mechanisms underpinning the mechanotransduction of hemodynamic forces by the vessel wall. The only significant criticism is that new insights derived from electrophysiologic and molecular studies have received little attention in this textbook. New contributions to the field will likely be derived from investigators in these fields. Nevertheless, this book provides the reader with an in-depth review of the physiologic studies that have contributed to our understanding of the mechanisms of mechanotransduction in the circulation.

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**Working with insurance and managed care plans: A guide for getting paid**

Jan Davison, Maxine Lewis. New York, 1995, McGraw-Hill, 448 pages, $39.95.

This book, aimed at the needs of medical office personnel, provides an introduction to current methods of reimbursement and the record keeping and billing practice they require. The contents are divided between two broad topics: the mechanics of billing and collecting, and an analysis of different forms of medical insurance. The first seven chapters provide extensive details on billing, with special attention to using the universal claim form. There are long chapters on diagnosis coding (ICD-9) and procedure coding (CPT), which provide good explanations of these topics. These sections would be a good introduction for any physician who is not clear about these topics. Most of the balance of the book describes different health care programs, including Medicare, Medicaid, private insurance programs, and the new directions in managed care. The emphasis is on the aspects of the programs related to filing claims and reimbursement policies. The chapter on Medicare provides a detailed account of calculation of reimbursement under RBRVS.

The main focus throughout the book is on how to prepare the paperwork to maximize reimbursement for the services provided while reducing to a minimum the frequency of rejected claims. Sample forms and examples from references such as the CPT and ICD-9 codes are extensively used. Part of the book is dedicated to aspects of office practices and record keeping beyond the actual preparation of claims. Numerous samples of office forms, sample letters, and patient materials are aimed at developing good record-keeping practices, which are increasingly important in this era of denied claims and postpayment audits. As an aid to learning, each chapter concludes with exercise section covering the concepts and data covered.

This book should provide a useful introduction to medical office staff who are going to be involved in claims preparation or any related record keeping. It may also be helpful reading for staffs of offices that have trouble with billing, especially if they have a high rate of denied claims. On the other hand, I do not consider this useful to the typical vascular surgeon either entering private practice after training or in an established practice. In this day and age the typical surgery resident has been exposed to the importance of documentation, the vagaries of reimbursement, and the use of coding systems. Most go into established practices. This book would be recommended as an overview for the occasional surgeon going into solo practice, for it will help him understand the importance of attention to well-established office practices.

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**L'Etoile du Nord: An account of Owen Harding Wangensteen**

Leonard Peltier, J. Bradley Aust. Chicago, 1994, American College of Surgeons, 158 pages, $20.

*L'Etoile du Nord* was produced by two authors, Leonard F. Peltier, MD, PhD, and J. Bradley Aust, MD, PhD, as a brief—very brief—account of the accomplishments of a truly remarkable surgeon, Owen H. Wangensteen. Both authors are former students of Dr. Wangensteen, as is this reviewer. We are also aware that a more detailed biography has been "in the works" for some time. The authors believed, however, that some recording of the life and influence of Dr. Wangensteen should be published now "so that he can be measured against his contemporaries, many of whose biographies are already in print." They have accomplished their stated goal in an interesting manner, frequently using Dr. Wangensteen's own published words to describe the situation and his role in it.

The first three chapters are about Dr. Wangensteen's early years and his education through medical school and surgical residency. Those chapters make two important points about Dr. Wangensteen. He was a voracious reader and relied heavily on the written word. He excelled in medical school, graduating first in his class. His surgical residency was unusual in that he spent a year with an internist, George E. Fahr, which he found to be a "wonderful opportunity." In 1923-24 Dr. Wangensteen was sent to the Mayo Clinic for a year of study. He seized on it as an opportunity to observe the premier physicians in the country and, as usual, he made the most of it, befriending William J. Mayo in the process.

The fourth chapter is about Dr. Wangensteen's travels in Europe, which had an enormous influence on his surgical thinking, particularly about resident education, taking clinical problems to the laboratory for investigation, and a